Pediatric laryngospasm: prevention and treatment

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Purpose of review

The purpose of this review is to discuss the risk factors associated with laryngospasm and the techniques used for prevention and treatment. We also summarize the prevention and treatment modalities in organized algorithms.

Recent findings

According to recent endoscopic studies, laryngospasm is always complete, thus airway management and intravenous therapy are indicated. Parental history of children having upper respiratory infection is associated with increased risk of laryngospasm. Anesthesia administered by a pediatric anesthesiologist is associated with lower incidence of laryngospasm. Intravenous anesthesia is associated with lower incidence of laryngospasm than inhalational anesthesia. In tracheal intubation, the use of muscle relaxants decreases laryngospasm. Deep laryngeal mask airway removal is associated with lower incidence of laryngospasm in sevoflurane or isoflurane anesthesia. In no intravenous line situation, laryngospasm can be treated with succinylcholine administration by intramuscular, intraosseous and intralingual routes.

Summary

Identifying the risk factors and taking the necessary precautions are the key points in prevention of laryngospasm. An experienced anesthesiologist is associated with lower incidence of laryngospasm. Airway management is the most essential part of treatment of laryngospasm. Drugs can be used as an adjunct in treatment of laryngospasm, especially when anesthesia is administered by beginners.

Keywords

airway, anesthesia, complication, laryngospasm, pediatric, prevention, treatment

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Introduction

Laryngospasm is a protective reflex closure of the glottis as a result of abnormal stimulus [1]. In a classic report, Fink [2] described two types of laryngospasm: expiratory stridor, which is an active closure of the glottis secondary to adductor spasm and inspiratory stridor, which is a passive closure of the glottis secondary to a ball–valve mechanism. If laryngospasm persists, it may cause hypoxia and hypercapnea. Most of the time, the resulting hypoxia abolishes the reflex and the spasm tends to be self-limited [2–4]. However, in rare occasions, serious morbidity such as cardiac arrest, arrhythmia, pulmonary edema, bronchospasm or aspiration may ensue [4–7].

Statistics

Laryngospasm causes about 40% of postextubation airway obstruction [8]. The incidence of laryngospasm in pediatric population ranges from 0.04 to 14% [4,5,9••, 10–12]. The incidence of complications resulting from laryngospasm can vary as follows: cardiac arrest 0.5%, obstructive negative pressure pulmonary edema 4%, pulmonary aspiration 3%, bradycardia 6% and oxygen desaturation 61% [13]. In a study [9••] characterizing the treatment interventions of laryngospasm, 38.1% of patients responded to mask ventilation with continuous positive airway pressure (CPAP) and 47.6% responded to muscle relaxants associated with ventilation.

Risk factors

Risk factors for laryngospasm can be classified into three categories: patient-related, surgery-related and anesthesia-related factors.

Anesthesia-related factors

The most important anesthesia-related risk factor is light level of anesthesia [14]. Extubating the trachea in patients while lightly anesthetized predisposes to laryngospasm. Any stimulation during a light plane of anesthesia such as pain, movement of the cervical spine and placement of nasogastric tube may predispose to laryngospasm. Also, vocal cord irritation by volatile anesthetics, secretions, mucus, blood, laryngoscopy blade and suction catheter may induce laryngospasm. Inexperience

of anesthesiologist can be associated with increased incidence of laryngospasm [15–17]. Multiple endotracheal intubations and laryngeal mask airway (LMA) insertion attempts increase the risk of laryngospasm [12,18,19].

There is controversy in the literature regarding the use of the airway device and associated risk of laryngospasm [20,21]. The endotracheal tube (ETT) was shown to be associated with increased incidence of laryngospasm [4]. The use of facemask in URI was suggested to be associated with low incidence of laryngospasm [22]. However, in three recent prospective studies [23–25], there was no statistical difference of the incidence of laryngospasm among facemask, LMA and ETT. This may have been attributed to beta error, too small a sample size for a rare occurrence. On the contrary, in two retrospective studies [9••,26••], LMA was shown to increase the incidence of laryngospasm. However, data collection accuracy and LMA's appropriate use in these studies have been questioned. It is suggested that the use of cuffed tracheal tubes in younger than 4-year-old children may predispose to laryngotracheal injury and laryngospasm [27,28•]. There are controversial reports about the preoperative use of midazolam and its effects on laryngospasm in literature. Although some studies showed an increased incidence of laryngospasm with preoperative use of midazolam, others [26••,29–31] have demonstrated the opposite. Midazolam combined with remifentanil has shown lower incidence of laryngospasm in comparison with halothane with fentanyl in children undergoing eye surgery under general anesthesia [28•]. Among the intravenous (i.v.) induction agents, thiopentone is associated with the highest risk of laryngospasm [4,6,10,32]. Intramuscular (i.m.) ketamine is associated with 0.4% incidence of laryngospasm. Furthermore, i.v. ketamine may indirectly predispose to laryngospasm by increasing secretions, which irritate the vocal cords [5,33,34]. Propofol decreases airway responsiveness secondary to depressing laryngeal reflexes [35,36]. Anesthesia induction with propofol is less associated with laryngospasm than induction with sevoflurane [37]. Among all the volatile anesthetics, desflurane has the highest incidence of laryngospasm (50%), followed by isoflurane, enflurane, halothane and sevoflurane, with no difference in the incidence of laryngospasm between the last two agents [6,38–40].

Patient-related factors

The most important patient-related risk factor is young age [4,10,17]. Although few studies [41,42] failed to show increased risk of laryngospasm in children with URI, most studies [17,26••,43] showed that URI increases the risk of laryngospasm from 2.3 to five-fold. The parental confirmation of a URI seems to be a better predictor of laryngospasm than the use of predetermined criteria [10]. Smoker adolescents are also prone to develop laryngospasm [44,45]. Passive smokers and children with hyperactive airway, including asthma, are 10 times more prone to develop laryngospasm [4,14,46] Other risk factors include American Society of Anesthesiologists (ASA) IV [26••], ex-premature under 1 year old, whooping cough, obstructive sleep apnea, obesity, airway anomaly, gastroesophageal reflux disease [26••,47–50], elongated uvula and history of choking during sleep [14,51,52]. Finally, electrolyte disturbance such as low magnesium and calcium blood level may predispose to laryngospasm [53].

Surgery-related factors

Among all surgery types, airway procedures, including bronchoscopy, have high incidence of laryngospasm. Tonsillectomy and adenoidectomy (T&A) surgery has an incidence of 21–27% [17,54–57]. Other types of surgery such as appendicectomy, hypospadias and transplant in children are associated with an increased risk of laryngospasm [4]. Esophageal endoscopy predisposes to laryngospasm secondary to stimulation of distal afferent esophageal nerves [58].

Prevention

Prevention of laryngospasm generally can be achieved by identifying and alleviating the risk factors when possible (Fig. 1).

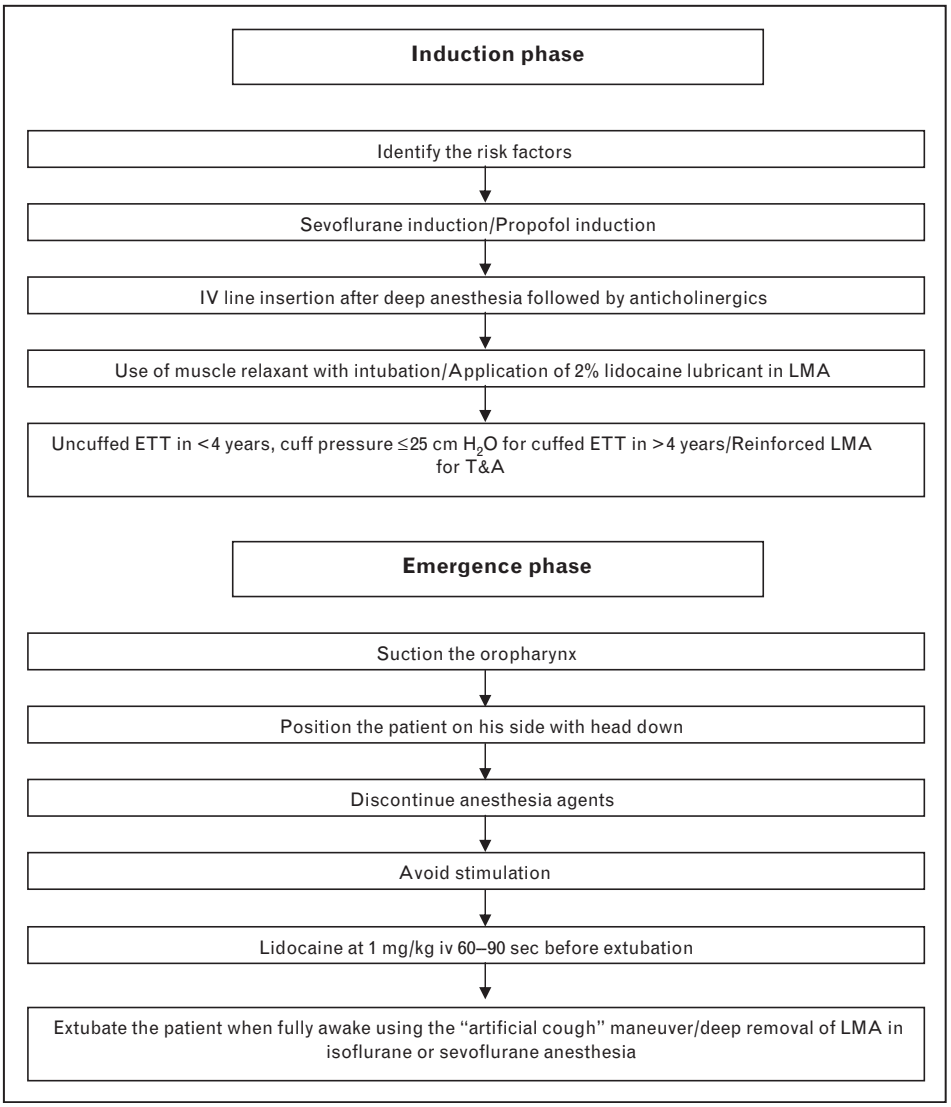
Prevention in preoperative phase

In the preoperative phase, a detailed history should be taken to identify the risk factors. Household exposure to smoking should be questioned. If an adolescent is a smoker, a period of abstinence from smoking of at least 48 h up to 10 days may be required to reduce the risk of laryngospasm [45]. In children with URI, the use of facemask may decrease the incidence of laryngospasm by minimizing airway irritation [22,59]. The debate regarding the optimal time to delay anesthesia to children following URI is still ongoing [60–62]. It usually takes 6–8 weeks for airway irritability to resolve after URI [63,64]; however, by that time, many children will have another URI and an increased risk of laryngospasm [65]. Thus, rather than postponing the surgery, it would be more beneficial to identify and alleviate the risk factors associated with URI that predict the respiratory complications. These factors are parent's statement that the child has URI, passive smoking, presence of nasal congestion or sputum, history of reactive airway disease, history of snoring, anesthetic agents, use of ETT and airway surgery [65,66].

Prevention during induction phase

To decrease the overall risk of laryngospasm, anesthesia should be carried out by an experienced anesthesiologist [10,17]. Although the effect of anticholinergic drugs is

Figure 1 An algorithm for prevention of laryngospasm



ETT, endotracheal tube; LMA, laryngeal mask airway; T&A, tonsillectomy and adenoidectomy.

controversial, these drugs may reduce the incidence of laryngospasm secondary to decreased secretions [6,57,67,68]. Early detection of laryngospasm can be achieved via continuous monitoring of respiratory sounds via precordial stethoscope [6]. Inhalational induction should be carried out by halothane or preferably sevoflurane. It is suggested that nitrous oxide (N_2O) should be avoided at induction to achieve higher oxygen reserve to prevent early desaturation in the event of laryngospasm [69]. Intravenous line insertion should be delayed until after deepening the anesthesia, with patient breathing regularly to decrease the incidence of laryngospasm [69,70]. The use of muscle relaxants concomitant with tracheal intubation reduces the risk of laryngospasm [17]. In anesthesia using LMA, the application of 2% lidocaine gel may decrease laryngospasm [71].

Prevention during maintenance phase

In patients less than 4 years of age, it is suggested to use uncuffed ETT to minimize the risk of laryngeal edema and laryngospasm [27,72]. If cuffed ETT are to be used, air leak at less than 25 cm water may decrease the incidence of laryngospasm [18,73]. In a prolonged surgery involving ETT, it is suggested to inflate the cuff with water to avoid N_2O diffusion and mucosal damage that predisposes to laryngospasm [73]. Whenever air is used, we suggest continuously monitoring the cuff pressure to avoid overinflation secondary to N_2O diffusion.

Prevention in emergence phase

Laryngospasm tends to occur more often during emergence than during induction of anesthesia [74]. It is still controversial whether the trachea should be extubated

deep or awake in order to decrease laryngospasm. Several studies [24,75–77] showed no difference in the incidence of laryngospasm between the two methods. Other studies [73,78–80] suggest that deep extubation reduces the incidence of laryngospasm. On the contrary, in a small sample size of patients, Tsui *et al.* [81] showed no single case of laryngospasm when performing awake extubation using the 'no touch technique' in T&A cases. This technique consists of suctioning the oropharynx, turning the patient to the lateral side with the head down to keep the vocal cords clear of secretions, discontinuing the anesthetics and avoiding stimulation until the patient becomes fully awake for extubation [81]. However, suctioning the oropharynx may not be adequate. An 'artificial cough' to decrease laryngospasm consists of positive-pressure inflation of the lungs just before extubation to expel the remaining secretions [19,82]. This technique may also reduce laryngospasm by decreasing the adductor response of the laryngeal muscles [78,83,84]. In anesthesia using LMA, there is a controversy regarding the timing of LMA removal to avoid laryngospasm. Some authors recommend awake removal of LMA in children [85–87]. However, in both sevoflurane and isoflurane anesthesia, there is a higher incidence of laryngospasm during awake compared with deep LMA removal [88–90]. Combining caudal block with isoflurane anesthesia resulted in similar incidence of laryngospasm between the two groups [91]. In halothane anesthesia, there is no difference in the incidence of laryngospasm between the two techniques [12,92–94].

Prevention in tonsillectomy and adenoidectomy

Among all surgery types, T&A has the highest incidence of laryngospasm (21–27%). Three studies [95–97] showed a statistically significant decrease in the incidence of laryngospasm in children undergoing T&A using armored LMA for airway management.

Prevention using drugs

Among all the drugs used to prevent laryngospasm, the most important and substantiated one is lidocaine. The beneficial effects of i.v. lidocaine may be attributed to a central increase in the depth of anesthesia. The first study was by Baraka [98] who administered i.v. lidocaine at 2 mg/kg 60–90 s before extubation in children undergoing T&A. However, Leicht *et al.* [99] underwent a similar investigation and found contradictory results; they attributed this to the different time interval of lidocaine administration (4.5 min) and claimed that lidocaine can be effective when trachea is extubated before signs of swallowing occur. Topical lidocaine at 4 mg/kg is equally effective in preventing laryngospasm in children and neonates [54,100]. Other drugs suggested for prevention are cocaine and magnesium [55,101].

Other techniques of prevention

Five percent CO_2, which still exists in very few anesthesia machines, can be given 5 min prior to extubation to prevent laryngospasm. This could be explained by the respiratory drive to exhale the CO_2 that overrides the spasm reflex [102]. Also, acupuncture decreased the incidence of laryngospasm to 5% [56].

Treatment

Treatment success mainly depends on the experience of the anesthesia provider. Removal of the offending stimulus alone may be sufficient to treat laryngospasm. Until recently, laryngospasm was classified into partial versus complete. The treatment was different for each type [103•]. However, recent reviews [104,105] derived from endoscopy studies showed that partial laryngospasm does not exist and laryngospasm is complete; thus, treatment should be the same (Fig. 2).

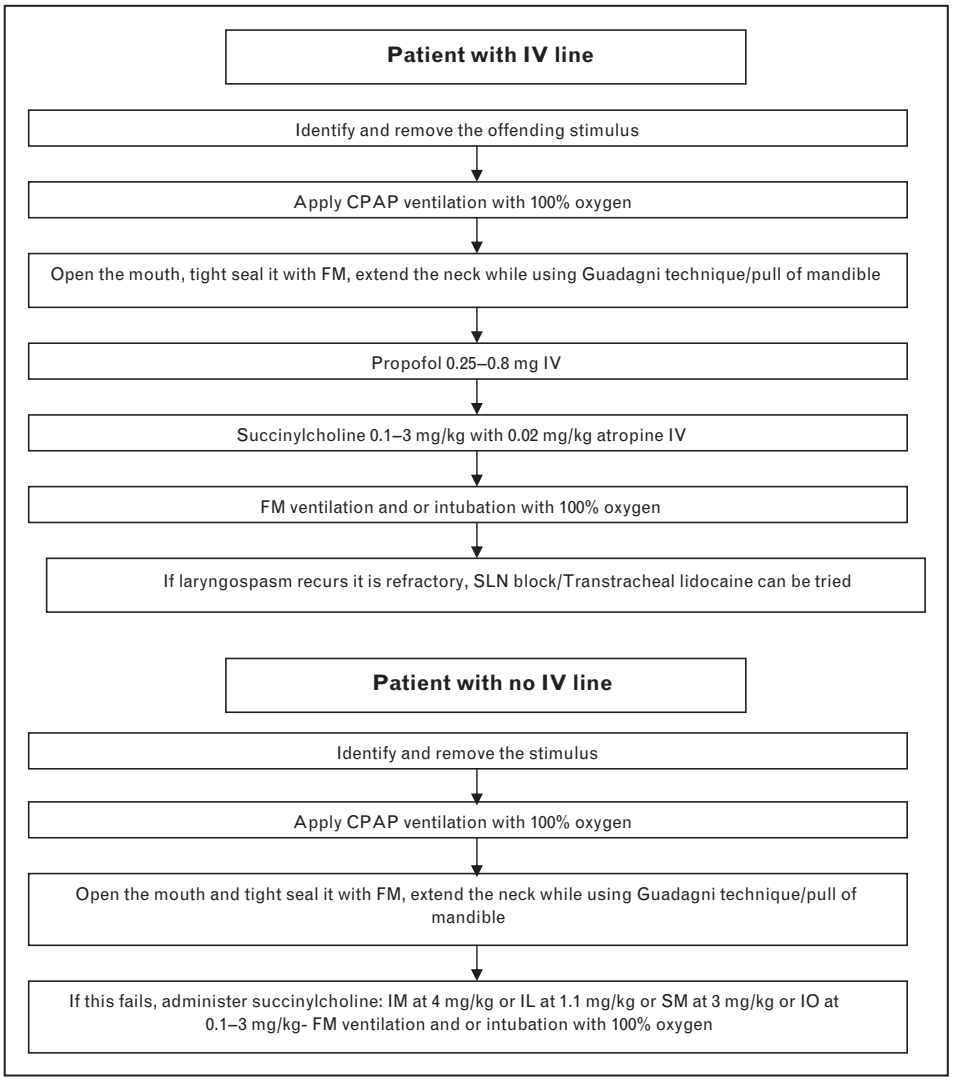
Treatment with airway management

Airway management includes opening the mouth, tight sealing with facemask, extending the neck with jaw lift and applying CPAP ventilation with 100% oxygen [106]. Airway management can be enhanced by two maneuvers. The first involves placing the middle finger of each hand in the laryngospasm notch located between the mastoid process and the ear lobule and pressing inward on the styloid process. This induces periosteal pain resulting in autonomic nervous system reflex and vocal cords relaxation [107,108]. The second maneuver consists of a vigorous forward pull of the mandible. This causes a painful stimulus and stretches the geniohyoid muscle to partially open the larynx [109]. Incomplete oxygenation secondary to stomach insufflation may occur during CPAP. An orogastric tube may be inserted for stomach deflation following the resolution of the spasm [110].

Treatment using drugs

Recent studies have shown that laryngospasm is always complete thus anesthesia with inhalational agents alone is not therapeutic. Rather, airway management and intravenous therapy is indicated. Propofol administered at 0.25–0.8 mg/kg i.v. can treat laryngospasm in 76.9% of cases. However, propofol is not studied in children less than 3-year-old [57,111–113]. Succinylcholine is still considered the gold standard for treatment of laryngospasm. It can be given at 0.1–3 mg/kg i.v. together with atropine at 0.02 mg/kg to avoid the possible succinylcholine-induced bradycardia and cardiac arrest [114]. Laryngospasm may recur after succinylcholine metabolism and a second dose may be given following atropine [14]. The use of a smaller dose of succinylcholine offers the advantage of the avoidance of bradycardia following repeated doses, while maintaining spontaneous breathing, which thus avoids further hypoxia [115].

Figure 2 An algorithm for treatment of laryngospasm



CPAP, continuous positive airway pressure; i.m., intramuscular; i.v., intravenous.

Treatment using other drugs

Alfentanil and meperidine may break laryngospasm especially if it is secondary to painful stimulus. Diazepam is reported to treat laryngospasm. Doxapram at 1.5 mg/kg i.v. can abolish laryngospasm by increasing the respiratory drive [15,31,116,117] Nitroglycerin 4 μg/kg i.v. was used for treatment of laryngospasm [118], although one can argue against nitroglycerine as it acts on smooth muscle, whereas the vocal cords consist of skeletal muscle. In addition to prevention, lidocaine can also play a role in treatment of laryngospasm secondary to airway suppressant effect [119].

Treatment when no intravenous access

If the patient has no i.v. access, the first-line treatment would be airway management using CPAP with 100% O_2. If laryngospasm persists, several approaches can be tried none of which is evidence based. In an editorial, Donati and Guay [120] recommended seeking help to establish i.v. access, including femoral vein for drug administration to treat laryngospasm. However, this may be time-consuming during this critical moment when patient needs immediate ventilation and oxygenation. Other authors advocate direct laryngoscopy and intubation with or without topical lidocaine [121]. However, this may prolong the hypoxic period and lead to significant airway trauma. Furthermore, topical lidocaine itself may induce laryngospasm [122]. In another editorial, Walker and Sutton [123] suggest treating laryngospasm with succinylcholine via one of the routes: i.m., intralingual, submental and intraosseous. The i.m. dose for succinylcholine is 4 mg/kg, the disadvantages being unpredictable absorption in near arresting patients and a rare occurrence of pulmonary edema. The intralingual dose is 1.1 mg/kg

the disadvantage being arrhythmias, the submental dose is 3 mg/kg and the intraosseous dose is the same as the i.v. They claim that obtaining sufficient paralysis for adequate ventilation and oxygenation can be achieved within 60 s by administering succinylcholine via one of the mentioned routes [124,125].

Treatment of refractory laryngospasm

If airway management and drug administration are tried and laryngospasm recurs, two techniques can be used. Superior laryngeal nerve (SLN), a branch of vagus nerve can be blocked to treat laryngospasm [15,126]. Transtracheal lidocaine injection through the cricothyroid membrane can also break the spasm [127]. However, these two techniques are described as case reports in adult patients.

Postlaryngospasm follow-up

Once laryngospasm is relieved humidified oxygen can be administered to decrease laryngeal irritation. Patients who experienced severe laryngospasm should be observed for 2–3 h to ensure that pulmonary complications are not overlooked [14].

Conclusion

Identifying the risk factors and taking the necessary precautions are key points in preventing laryngospasm. To overall decrease the risk of laryngospasm, anesthesia should be carried out by experienced anesthesiologist. The timing of extubation in order to decrease laryngospasm still needs to be answered. However, awake extubation using the 'no touch technique' may be promising but needs further studies. LMA removal should be carried out at deep level of anesthesia when using isoflurane or sevoflurane. The relation of the use of airway devices and the risk of laryngospasm needs to be further studied. Regarding management, in most cases, airway maneuver by an experienced anesthesiologist usually treats laryngospasm. However, drug administration may be needed to break the spasm especially in junior anesthesiologists.

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Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 452).

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